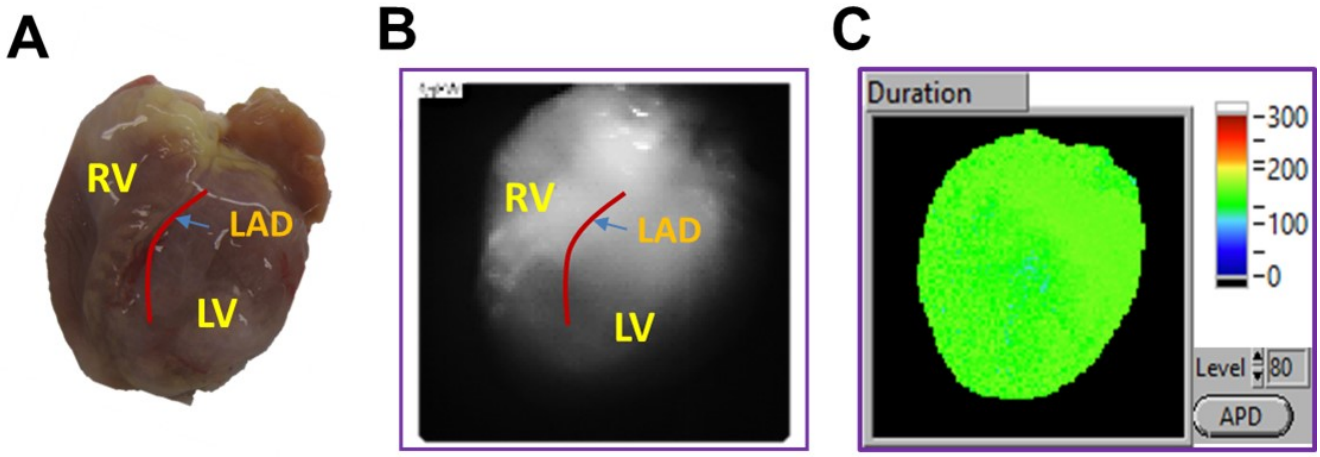
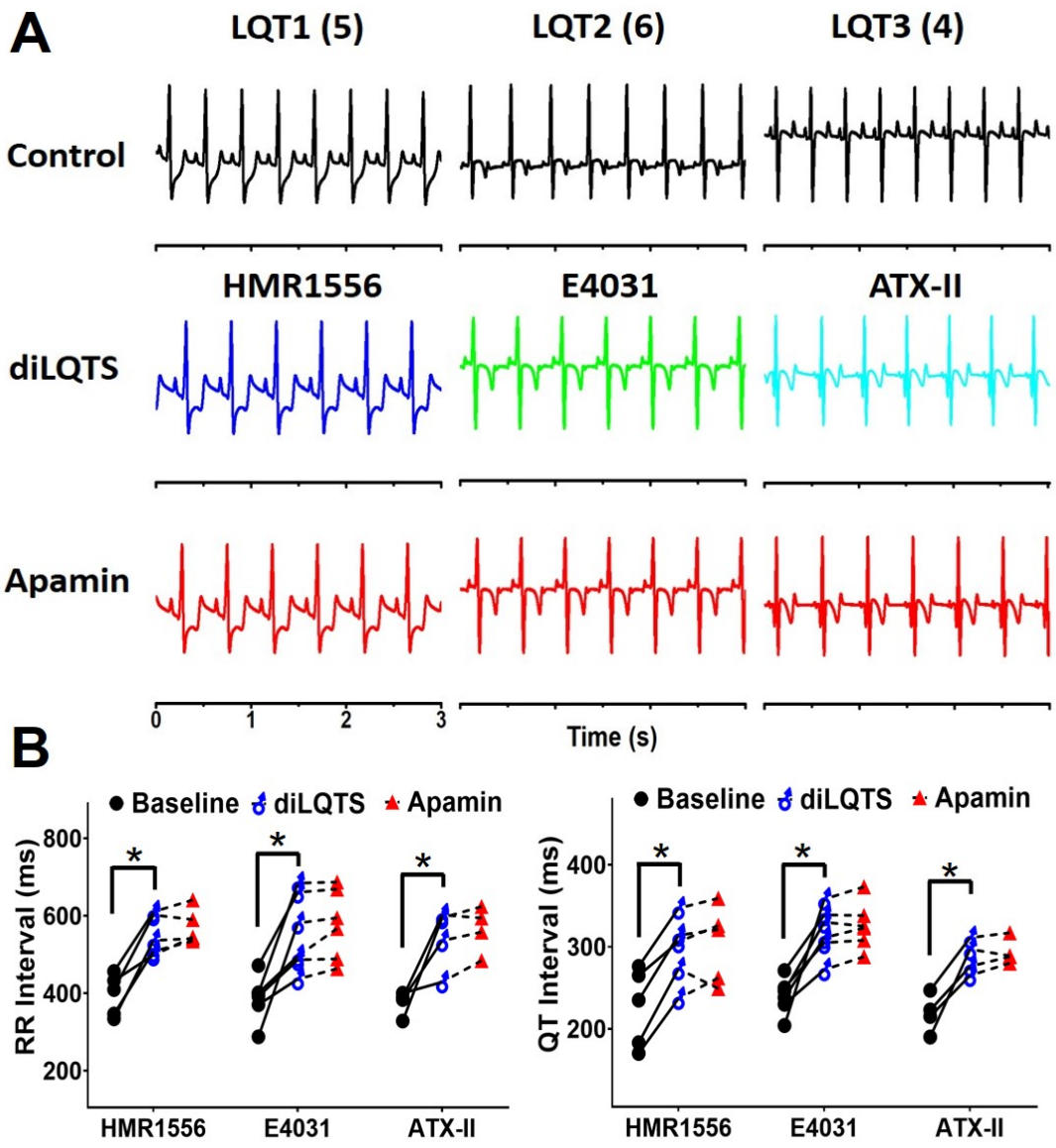
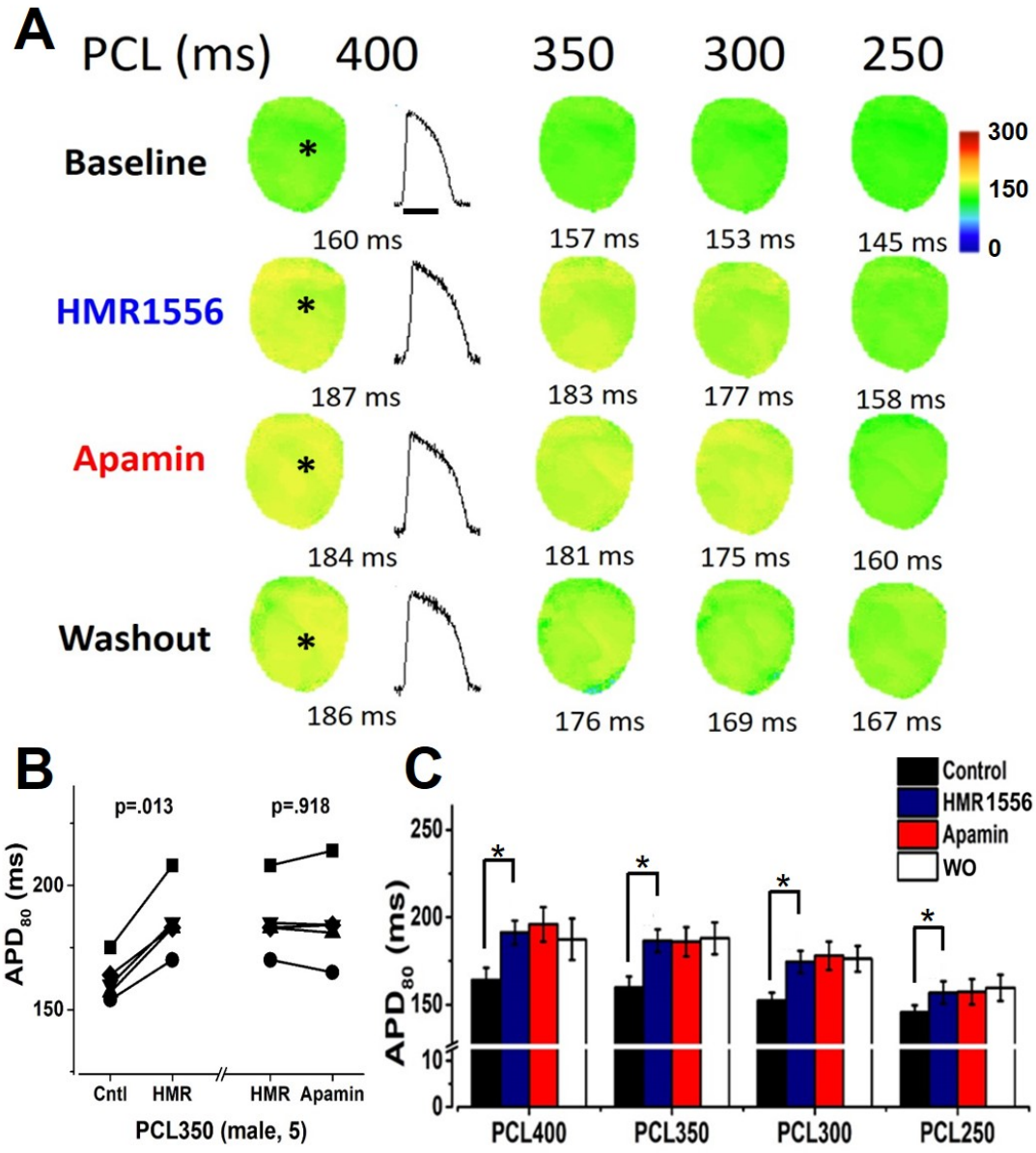


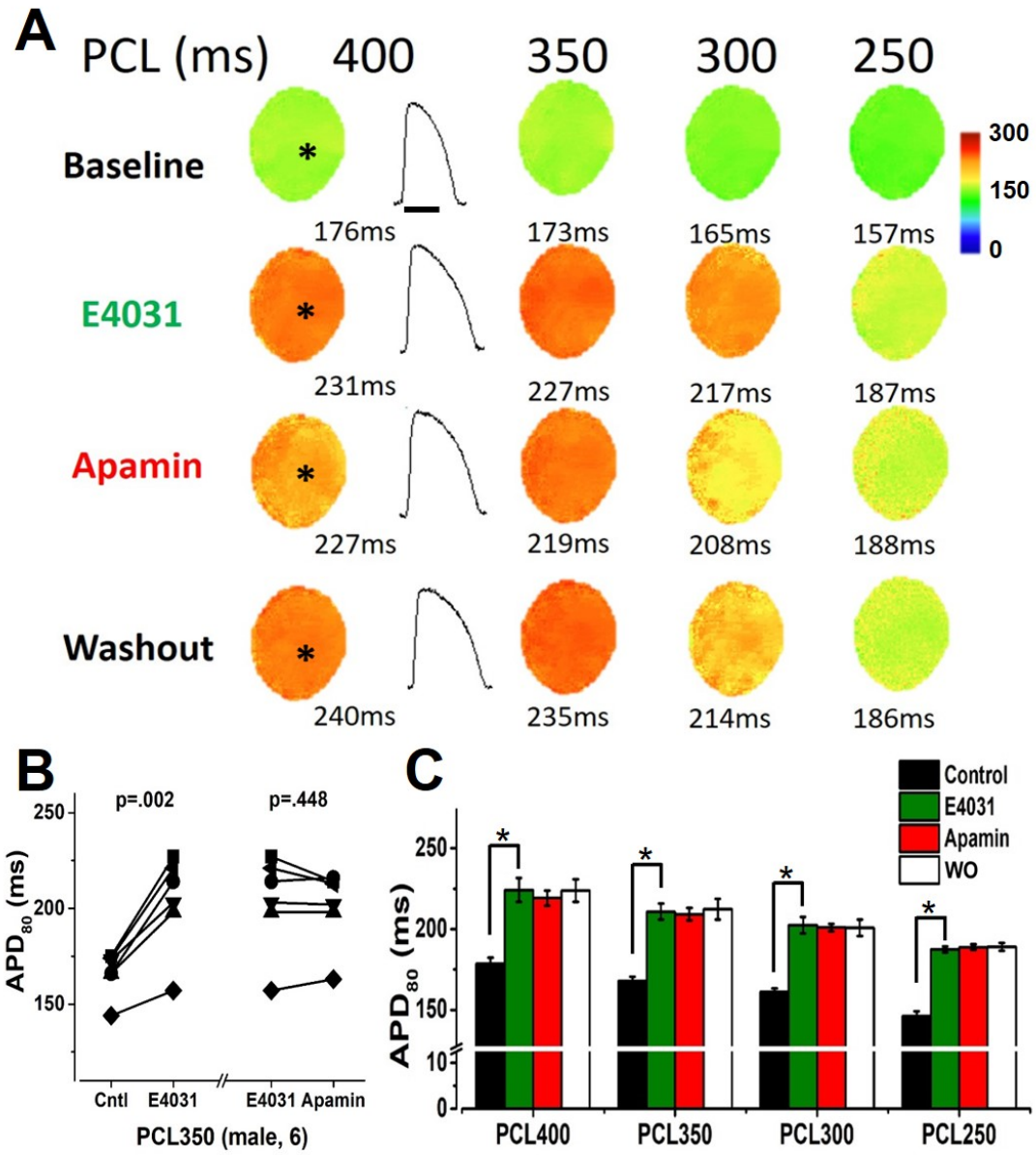
Online Supplement Figure 1. Diagram of an *ex vivo* intact heart seen by optical mapping camera. **A**, relative anatomical locations of the right ventricle (RV), the left ventricle (LV) and the left anterior descending artery (LAD). **B**, the preparation under the camera. **C**, an example of APD_{80} map in the region of interest. The APD or Ca_iTD are derived from the average value in the region of interest.



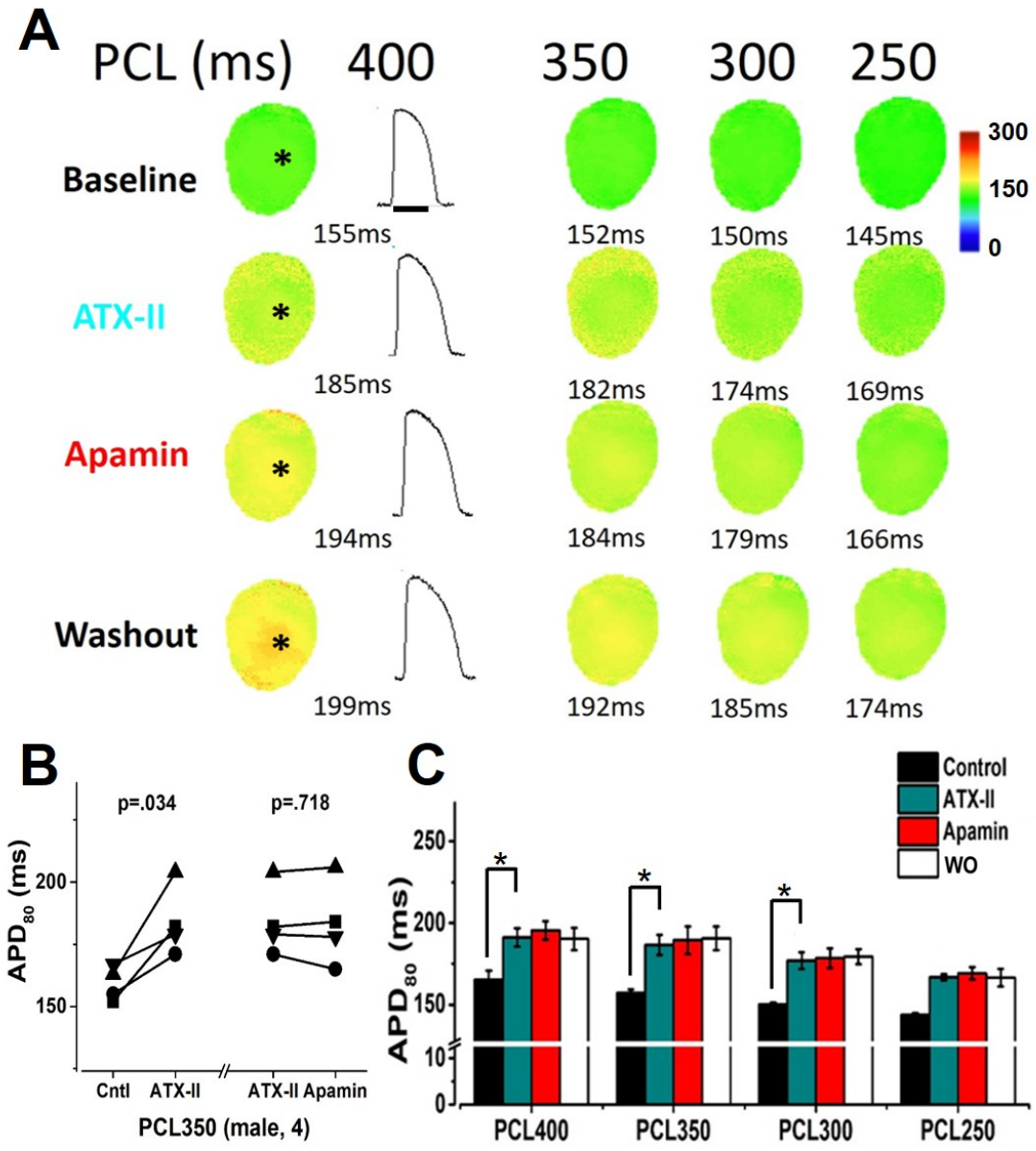
Online Supplement Figure 2. Apamin does not further prolong RR and QT intervals in male hearts with diLQTS. We included for analyses male hearts that maintained stable normal sinus rhythm throughout the study. **A** shows representative pseudo ECG traces of diLQT1 (HMR1556, 100 nmol/L), diLQT2 (E4031, 50 nmol/L), and diLQT 3 (ATX-II, 20 nmol/L). Apamin (100 nmol/L) was then given during continued drug infusion. **B,** linkage graphs show the changes of RR and QT intervals in different types of diLQTS. Asterisks indicate significant differences (*diLQTS compared with baseline; $p<0.05$, each n=4-6).



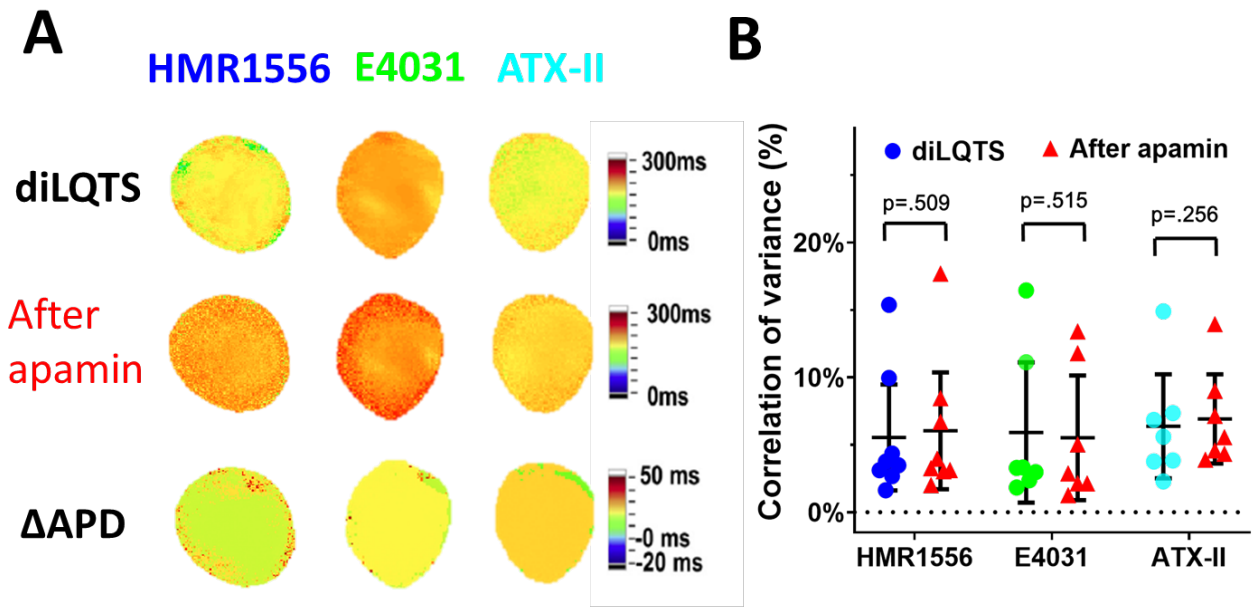
Online Supplement Figure 3. Apamin does not further prolong APD_{80} in male hearts pretreated with HMR1556. **A**, representative membrane potential traces and APD_{80} maps at baseline and in the presence of HMR1556 (100 nmol/L), after apamin (100 nmol/L), and after washout (Protocol I, diLQT1). **B,** at 350 ms PCL, HMR1556 slightly prolonged APD_{80} but no further increase of APD_{80} was observed after apamin. **C,** summary of apamin effects on APD_{80} at different PCLs in male rabbit ventricles. The apamin effects were not observed at any PCLs. Data presented as mean ± SEM. Asterisks indicate significant differences (*diLQTS compared with baseline; $p<0.05$, n=5).



Online supplement Figure 4. Apamin does not further prolong APD_{80} in male hearts pretreated with E4031. **A,** representative membrane potential traces and APD_{80} maps at baseline and in the presence of E4031 (50 nmol/L), after apamin (100 nmol/L) and after washout (Protocol II, diLQT2). **B,** at 350 ms PCL, E4031 (50 nmol/L) significantly prolonged APD_{80} but apamin did not cause further APD_{80} prolongation. **C,** summary of apamin effects on APD_{80} at different PCLs in male rabbit ventricles. The apamin effects were not observed at any PCLs. Data presented as mean ± SEM. Asterisks indicate significant differences (*diLQTS compared with baseline; $p<0.05$, n=6).



Online Supplement Figure 5. Apamin does not further prolong APD_{80} in male hearts pretreated with ATXII. **A,** representative membrane potential traces and APD_{80} maps at baseline and in the presence of ATX-II (20 nmol/L), after apamin (100 nmol/L), and after washout (Protocol III, diLQT3). **B,** at 350 ms PCL, ATX-II significantly prolonged APD_{80} but without further increased APD_{80} by apamin. **C,** summary of apamin effects on APD_{80} at different PCLs in male rabbit ventricles. The apamin effects were not observed at any PCLs. Data presented as mean ± SEM. Asterisks indicate significant differences (*diLQTS compared with baseline; $p<0.05$, n=4).



Online Supplement Figure 6. Effects of apamin on action potential duration (APD) heterogeneity at PCL350 ms in female diLQTS ventricles. A, representative APD_{80} maps at three diLQTS and after adding of apamin (100 nmol/L). The delta APD maps showed no large heterogeneities on the epicardium. B, apamin had no significant effects on the correlation of variance of APD_{80} in female diLQTS ventricles.